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Figure 1. A) Regulatory T cell mechanisms of suppression. Regulatory T cell (Treg) can suppress by four basic mechanisms. The interaction between cytotoxic T lymphocyte antigen-4 (CTLA4) and CD80/CD86, expressed by antigen presenting cells (APCs), leads to CD80/CD86 down-regulation. Removal of these co-stimulatory molecules modulates APC function, limiting the initiation of an adaptive immune response. Tregs induce effector T cell (Teff) apoptosis by the interaction between Galectin-9 (Gal-9) and the T cell immunoglobulin and mucin domain-3 (TIM-3), and by the release of granzymes which enter Teffs via perforin pores. Tregs release the anti-inflammatory cytokines TGFβ, IL10 and IL35. Treg expression of the ecto-enzymes CD39 and CD73 enables the hydrolysis of pro-inflammatory adenosine triphosphate (ATP) into anti-inflammatory adenosine (ADO). **B) Regulatory T cell defects in autoimmunity.** In health, Tregs maintain tolerance by exerting suppression of effector T cells. In organ specific autoimmune disease, Tregs fail to suppress autoreactive effector T cells, therefore leading to target cell death. Reported reasons for this include inadequate numbers of Tregs, impaired suppressive ability, Treg conversion into effector cells and resistance of effector T cells to Treg-mediated suppression.